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Considering the complexity of microbial community dynamics in food safety risk assessment☆

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Abstract

The potential for competitive inhibition to limit the growth of microbial pathogens in food raises questions about the external validity of typical predictive microbiology studies and suggests the need to consider microbial community dynamics in food safety risk assessment. Ecological theory indicates, however, that community dynamics are highly complex and may be very sensitive to initial conditions and random variation. Seemingly incongruous empirical results for *Escherichia coli* O157:H7 in ground beef are shown to be consistent with a simple theoretical model of interspecific competition. A potential means of incorporating community-level microbial dynamics into the food safety risk assessment process is explored.
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1. Introduction

Predictive microbiology has made significant contributions to food safety risk assessment and risk management (McMeekin et al., 1997). Like the results of other experimental studies, however, nagging concerns remain about the external validity of predictive microbiology models for drawing inferences about real world exposures to microbial pathogens in food. Typically, such models have been developed on the basis of monospecific cultures grown in an artificial matrix under static abiotic environmental conditions. Predictive microbiological models typically have failed to account for non-steady-state environmental conditions and diversity in the physiologic status of microorganisms and pretreatment storage conditions (McMeekin et al., 1997). Furthermore, pathogen growth rates and maximum densities are thought to be a function of the total microbial community composition and density in the food due to competition for nutrients, the production of inhibitory substances, and overall density. Thus, the potential for spoilage and

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other normal food microflora to competitively inhibit the growth of pathogens also raises questions about the validity of monospecific culture experimental results with respect to growth rates, maximum population density (MPD), and other aspects of pathogen population dynamics in naturally contaminated food products. Competitive inhibition of foodborne pathogens has been demonstrated for *Salmonella*, where the suppression of growth of all microorganisms occurred when the total microbial population density achieved the upper limit characteristic of the growth matrix (Jameson, 1962). This effect has also been reported for *Staphylococcus aureus*, *Listeria monocytogenes*, *Yersinia enterocolitica*, *Bacillius cereus*, *Salmonella infantis*, and *Carnobacterium* spp. (Buchanan and Bagi, 1997; Duffes et al., 1999; Grau and Vanderlinde, 1992; Mattila-Sandholm and Skytta, 1991; Nilsson et al., 1999; Ross and McMeekin, 1991). The observed dynamics of mixed microbial populations can be highly complex, however. Buchanan and Bagi (1999) demonstrated, for example, that *L. monocytogenes* grown in co-culture with *Pseudomonas fluorescens* can attain maximum population densities that are lower, higher, or the same compared to levels of the pathogen monoculture, depending on the temperature, acidity, and availability of water in the surrounding environment. Such results indicate that fully considering the complexity of microbial community dynamics would require detailed knowledge of the food, its microbial composition and inoculum levels, the factors affecting competitive interactions, and how the food is handled during transportation, storage, distribution, and use.

Undoubtedly, predictive microbiological models based on multi-species trials would present a more realistic picture of microbial community dynamics in food products. As a practical matter, however, an experimental program that evaluates all possible combinations of abiotic and biotic environmental conditions would be prohibitively costly and time-consuming. There are also practical limits on the successful identification and enumeration of target organisms and their competitors given currently available microbiological selective culture methods. Therefore, great care in experimental design will be needed to ensure that the value of information provided by community-level studies warrants the time and resources allocated to them. From an experimental perspective, judicious use of theoretical ecology models has the potential to inform efficient community-level study design by helping to identify important regions in the experimental design space (e.g., the growth/no-growth interface). Furthermore, theoretical modeling can help to construct general explanations for specific observed results. This application of theoretical modeling can be particularly useful when results are unexpected. Therefore—as a practical matter—the insights gained from theoretical ecology may help to avert potentially unproductive disagreements arising from seemingly contradictory empirical results.

One apparent contradiction arises from the intuitive notion that ubiquitous natural spoilage flora (e.g., *Pseudomonas* species) will inevitably outcompete and eventually exclude comparatively rare pathogens in food products. Responses observed under experimental conditions vary considerably, however (e.g., Buchanan and Bagi, 1999). Further, theoretical ecology indicates that the course of competitive interactions between microorganisms may be substantially altered or even reversed due to variation among strains or environmental conditions or as a consequence of chance events, such as differences in initial concentrations between pathogens and other microflora within the food substrate. This paper first illustrates the consistency of seemingly incongruous results from predictive microbiological experiments with a simple model of interspecific competition, using *Escherichia coli* O157:H7 in ground beef as an illustrative example. We then explore how community-level microbial dynamics could be incorporated into the food safety risk assessment process.

1.1. Predictive microbiology for E. coli O157:H7

Predictive microbiology models have been developed for *E. coli* O157:H7 under a variety of environmental conditions. These models predict the growth and decline of *E. coli* O157:H7 given environmental parameters including time, temperature, pH, and salinity. One set of equations was developed by Buchanan and Bagi (1994) based on studies of monospecific cultures grown in brain heart infusion broth. This set of equations was later incorporated into the Pathogen Modeling Program

(PMP) available from the US Department of Agriculture, Agricultural Research Service (ARS). Based on the ARS data, Marks et al. (1998) calculated the maximum population density (e.g., the observed maximum number of *E. coli* O157:H7 colony-forming units per gram (cfu/g)) as a function of the theoretical maximum density (TMD) and temperature. Marks et al. (1998) estimated the TMD of *E. coli* O157:H7 at refrigeration temperatures to be about 10 log (10^{10} cfu/g).

Walls and Scott (1996) compared predictions from the PMP with observations of *E. coli* O157:H7 growth in ground beef with natural flora and concluded that the PMP "offers reasonably good predictions of growth in raw ground beef". In particular, Walls and Scott (1996) demonstrated growth in ground beef up to approximately 10 log. (Note that the figures in Walls and Scott (1996) present the average levels for the experimental replicates.) How is it possible that the MPD of *E. coli* O157:H7 co-cultured with the natural ground beef flora could approach the theoretical maximum? Initially, the experimental results for *E. coli* O157:H7 cultured in raw ground beef appear to contradict those reported for *Salmonella*, *Listeria*, and other pathogens co-cultured with natural foodborne microflora. These seemingly paradoxical experimental results are consistent, however, with the complex range of outcomes predicted by a simple model of interspecific competition.

2. Methods

2.1. Lotka–Volterra competition model

The Lotka–Volterra competition model provides a basic model for the population growth of two interacting species (Brown and Rothery, 1993). The approach is an extension of the logistic model for population growth of a single species limited by a maximum carrying capacity characteristic of a particular habitat. The monospecific logistic growth model describes a limited population growth rate that decreases linearly with population density due intraspecific competition. This basic approach is extended to account for competition between species by incorporating an additional reduction in the population growth rate which is proportional to the population density of another species. The Lotka–Volterra model for two-species competition can be expressed as follows:

$$\frac{dN_1}{dt} = r_1 N_1 \left[1 - \frac{N_1 + \alpha_{12} N_2}{TMD_1}\right],$$
$$\frac{dN_2}{dt} = r_2 N_2 \left[1 - \frac{N_2 + \alpha_{21} N_1}{TMD_2}\right] \quad (1)$$

where N_1 and N_2 are the population densities of species 1 and 2, r_1 and r_2 are the intrinsic (unlimited) growth rates, TMD_1 and TMD_2 are the theoretically maximum population densities under monospecific growth conditions, and α_{12} and α_{21} are the interspecific competition coefficients, where α_{ij} refers to the competitive effect on species i by species j.

Dens et al. (1999) extends the basic Lotka–Volterra model to account for a lag phase prior to the onset of exponential growth:

$$\frac{dN_1}{dt} = r_1 N_1 \frac{Q_1}{1+Q_1} \left[1 - \frac{N_1 + \alpha_{12} N_2}{TMD_1}\right],$$
$$\frac{dN_2}{dt} = r_2 N_2 \frac{Q_2}{1+Q_2} \left[1 - \frac{N_2 + \alpha_{21} N_1}{TMD_2}\right],$$
$$\frac{dQ_i}{dt} = r_i Q_i \quad (2)$$

where Q_i, which represents the physiological state of the cells, grows exponentially and allows for description of the lag phase. Note that as $\alpha_{ij} \to 0$, Eq. (2) reduces to the conventional single species growth model presented by Baranyi and Roberts (1994). Further, as $Q_i \to \infty$, Eq. (2) reduces to Eq. (1), and as both $Q_i \to \infty$ and $\alpha_{ij} \to 0$, Eq. (2) reduces to the simple logistic growth model.

Lachowicz et al. (1995) provides an example of the successful application of the basic Lotka–Volterra competition model to describe the community dynamics of two *Shigella flexneri* strains (*S. flexneri* 3b, a mutant strain that carries a prophage lethal for the original strain, *S. flexneri* 1b) grown in co-culture. In particular, Lachowicz et al. (1995) found that analysis of serial cultivations suggests that empirical estimates of the Lotka–Volterra equation parameters are realizations (observed values) of random variables characterizing strains and media. This finding is noteworthy in that it introduces the key concept that

the microbial community dynamics are probabilistic—not deterministic, as implied by Eqs. (1) and (2). The implications of this additional layer of complexity become apparent in Results.

2.2. Competition scenario simulation

As a complement to fitting the Lotka–Volterra competition model to experimental data, population density time series can be simulated for scenarios consisting of specified values of the model parameters and initial population densities. For illustrative purposes, consider a simple, two-species microbial community consisting of a pathogen (species 1) and a spoilage organism (species 2). The basic Lotka–Volterra model (Eq. (1)) is used (i.e., lag phase dynamics are ignored). Table 1 summarizes the Lotka–Volterra competition model parameters for the scenarios considered in this paper.

Scenarios 1–3 assume that the intrinsic growth rate of both species 1 and 2 is equivalent to a 3-h generation (doubling) time (i.e., $r_1 = r_2 = 0.23$). By comparison, Walls and Scott (1996) reported generation times of 0.4–6.0 h for *E. coli* O157:H7 under various experimental growth conditions (12–35 °C, pH 5.7–6.4). Scenarios 1–3 also assume that the TMD of both species in ground beef is 10^{10} cfu/g (i.e., $TMD_1 = TMD_2 = 10$ log), and that the competitive effect of the spoilage organism on the pathogen is twice that of the pathogen's effect on the spoilage organism (i.e., $\alpha_{12} = 2$; $\alpha_{21} = 1$).

Scenarios 1–3 differ only with respect to the initial densities of the pathogen. In Scenario 1, the initial densities of the pathogen and spoilage organism (N_{10} and N_{20}, respectively) are both set at 10^2 cfu/g. In Scenarios 2 and 3, the initial spoilage organism density is held constant at $N_{20} = 10^2$ cfu/g while the initial pathogen density (N_{10}) is increased to 10^3 and 10^4 cfu/g, respectively. By comparison, Walls and Scott (1996) inoculated their ground beef with 10^3–10^4 cfu/g of *E. coli* O157:H7.

For a given scenario, we can evaluate what changes in a single competition model parameter would be required to achieve a specified outcome by holding all other factors constant and solving numerically for the remaining model parameter. For example, in Scenario 4, we seek to identify conditions that inhibit pathogen population growth such that the maximum density achieved (MPD_1) is 10^5 cfu/g. Scenario 4 assumes a base case as follows: $r_1 = r_2 = 0.23$ (3-h generation times); $TMD_1 = TMD_2 = 10$ log; $\alpha_{12} = 2$, $\alpha_{21} = 1$; and $N_{10} = N_{20} = 10^2$ cfu/g. While holding other factors constant, each competition model parameter is varied until $MPD_1 = 10^5$ cfu/g. Numerical solutions were performed using Microsoft© Excel™ ('97) loaded with the Solver add-in tool.

Scenarios 1–4 illustrate the effects of varying individual model input values on complex microbial community dynamics. This is the simple, deterministic form of the competition model (i.e., stochastic variation in growth rates and other model parameters has been ignored). Next, we consider the effect of varying the growth rates stochastically while holding other factors constant. Scenario 5 assumes that the growth rates of both species (r_1 and r_2) vary between 1- and 3-h generation times, depending upon environmental conditions (i.e., $r_i \sim$ uniform(0.23,0.68)). Scenario 5 further assumes that due to similar responses to temperature and other factors, r_1 and r_2 are highly, but not perfectly correlated (i.e., the linear correlation (r^2) between the growth rates is 90%). As in Scenario 1, let $TMD_1 = TMD_2 = 10$ log; $\alpha_{12} = 2$ and $\alpha_{21} = 1$; and $N_{10} = N_{20} = 10^2$ cfu/g. By employing Monte Carlo simulation methods (Vose, 2000), we can estimate

Table 1
Competition model parameters

Scenario	N_{10} (log cfu/g)	N_{20} (log cfu/g)	r_1	r_2	r^2 ($r_1 r_2$)	α_{12}	α_{21}	TMD (log cfu/g)
1	2	2	0.23	0.23	–	2	1	10
2	3	2	0.23	0.23	–	2	1	10
3	4	2	0.23	0.23	–	2	1	10
4—N_{20} changed	2	2.8×10^6	0.23	0.23	–	2	1	TMD = 10, $MPD_1 = 5$
4—r_2 changed	2	2	0.23	0.68	–	2	1	TMD = 10, $MPD_1 = 5$
4—α_{12} changed	2	2	0.23	0.23	–	4.3×10^4	1	TMD = 10, $MPD_1 = 5$
5	2	2	~ uniform(0.23,0.68)		0.90	2	1	10

the relative frequency of different competitive outcomes. Monte Carlo simulation procedures were performed with Latin Hypercube sampling (10,000 iterations) using Palisades© @Risk™ (Ver. 3.5.2), an add-on to Microsoft© Excel™ ('97).

2.3. Integrating the competition model into risk assessment

Next, we explore how community-level microbial dynamics could be integrated into the food safety risk assessment process. For illustrative purposes, we do so by inserting the simple Lotka–Volterra competition model into a process risk model for *E. coli* O157:H7 at the point of ground beef fabrication. We then proceed to expand on the previous scenarios and carry them forward to the risk assessment endpoints of ingested dose and the probability of human illness. Assuming the pathogen (species 1) to be *E. coli* O157:H7, consider again Scenarios 1–3, which differ only with respect to the initial concentration of the pathogen. Assume that 48 h of microbial community growth occurs prior to cooking. (The duration of conditions permitting growth need not be continuous, and we assume no net growth or mortality during no-growth conditions.) Assume further that cooking results in a 6-log reduction in microbial levels and that consumption consists of a 100 g serving (i.e., the ingested dose is 2 log higher than post-cooking density, which is expressed on a per gram basis).

In order to simulate the probability of illness from an ingested dose of *E. coli* O157:H7, a suitable dose–response function is required. For illustrative purposes, we employ a dose–response relationship for *E. coli* O157:H7 presented by Powell et al. (2000), which consists of the following beta-Poisson model:

$$p = 1 - \left(1 + \frac{d}{b}\right)^{-a} \tag{3}$$

where p is the probability of illness, d is the average administered dose, $a = 0.221$, and $b = 8722.48$. As indicated by Powell et al. (2000), however, this curve only represents a provisional estimate of the most likely value (MLV) of the dose–response function for *E. coli* O157:H7 within a broad range of uncertainty about the true dose–response function. (See Food Safety and Inspection Service (2001) for an alternative most likely value of the dose–response function for *E. coli* O157:H7.)

3. Results

3.1. Competition scenario simulation

The dynamics of simulation Scenarios 1–3 are presented in Figs. 1–3, respectively. The results illustrate how the competitive interaction simulation depends on the initial concentrations of the microorganisms. Note that Scenarios 1 and 2 (Figs. 1 and 2) reach the same competitive outcome—eventual exclusion of the pathogen by the spoilage organism—but they differ in the path taken. In Scenario 1 (Fig. 1), the maximum pathogen density achieved is reduced an order of magnitude below its theoretical potential. In Scenario 2 (Fig. 2), however, the pathogen density virtually achieves the TMD before being overtaken by the spoilage organism. (Similarly, Dens et al. (1999) demonstrated how consideration of lag phase dynamics (Eq. (2)) may result in the same competitive outcome as that predicted by the basic Lotka–Volterra model (Eq. (1)) but by means of a different course with respect to time.) In contrast, Scenario 3 (Fig. 3) results in a qualitatively different competitive outcome—coexistence characterized by dominance of the spoilage organism by the pathogen throughout the modeled time series.

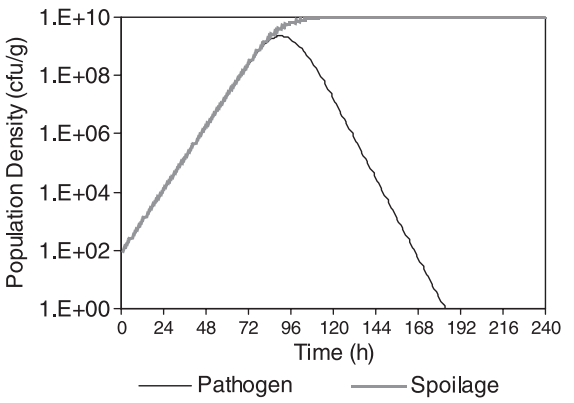

Fig. 1. Competition model with initial concentrations: $N_{10} = N_{20} = 1e+2$.

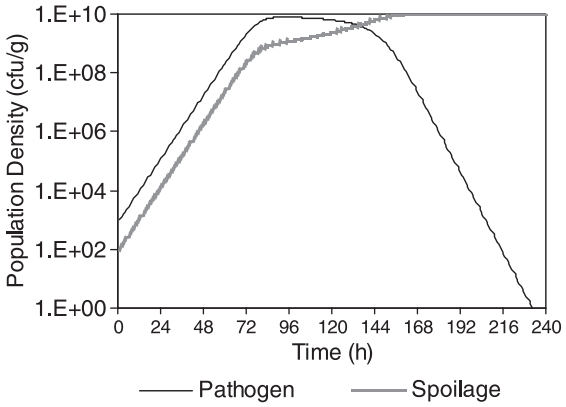

Fig. 2. Competition model with initial concentrations: $N_{10} = 1e+3$; $N_{20} = 1e+2$.

For Scenario 4, if while holding other factors constant, N_{20} is increased to 2.8×10^6, then MPD_1 is held to 10^5 cfu/g. Alternatively, if r_2 is increased to 0.68 (effectively reducing the generation time for the spoilage organisms to 1 h) while holding other factors constant, then MPD_1 is similarly inhibited. Also, if α_{12} is increased to 4.3×10^4 while holding other factors constant, then MPD_1 is held to 10^5 cfu/g (see Table 1) Thus under this scenario, limiting the maximum pathogen density to 5 log is more sensitive to changes in the spoilage organism growth rate than to changes in either the spoilage organism's initial density or the competitive effect of the spoilage organism on the pathogen. Note that with respect to the growth rate

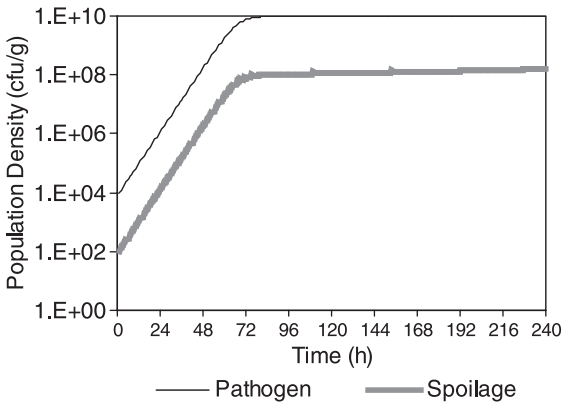

Fig. 3. Competition model with initial concentrations: $N_{10} = 1e+4$; $N_{20} = 1e+2$.

solution, the simulation indicates that the spoilage organism experiences a 1-h generation time under the same environmental conditions at which the pathogen grows at a 3-h generation time.

Under Scenario 5, the growth rates (r_1 and r_2) vary stochastically in a correlated fashion. Monte Carlo simulation results of Scenario 5 indicate that although the likelihood that the spoilage organism eventually excludes the pathogen is estimated to be 99%, the likelihood that the maximum pathogen density (MPD_1) achieved during the time series exceeds 10^9 cfu/g is estimated to be 80%.

3.2. Integrating the competition model into risk assessment

Table 2 summarizes the hypothetical risk estimates resulting from integrating the simple Lotka–Volterra competition model into the process risk model for *E. coli* O157:H7 under the scenario simulations. By simulating Scenarios 1–3 from the initial contamination levels of raw ground beef through the dose–response relationship (Eq. (3)), we can evaluate the impact of varying just the initial pathogen concentration on the selected risk assessment endpoints. For Scenario 1 ($N_{10} = N_{20} = 10^2$ cfu/g), the simulated ingested dose of *E. coli* O157:H7 at 48 h is 2.2 log cfu, and the estimated probability of illness is 0.004. For Scenario 2 ($N_{10} = 10^3$ cfu/g and $N_{20} = 10^2$ cfu/g), the simulated ingested dose of *E. coli* O157:H7 at 48 h is 3.2 log cfu, and the estimated probability of illness is 0.04. For Scenario 3 ($N_{10} = 10^4$ cfu/g and $N_{20} = 10^2$ cfu/g), the simulated ingested dose of *E. coli* O157:H7 at 48 h is 4.2 log cfu, and the estimated probability of illness is 0.21. Under this simple set of scenarios, a 2-log increase in N_{10} increases the probability of illness

Table 2
Hypothetical risk estimates

Scenario	Ingested dose (log cfu) at 48 h	Probability of illness
1	2.2	0.004
2	3.2	0.04
3	4.2	0.21
4	min = 0.0, max = 0.7	min = 0, max = 0.0001
5	median = 4.2, 95% CI = 2.1–5.8	median = 0.2, 95% CI = 0.003–0.62

by a factor of approximately 50 (less than two orders of magnitude).

For Scenario 4, in which MPD_1 is limited to 5 log by varying one competition model parameter at a time, the simulated probability of illness ranges from 0 (for $\alpha_{12} = 4.3 \times 10^4$, the pathogen is completely excluded prior to 48 h of microbial community growth) to 1×10^{-4} (for $N_{20} = 2.8 \times 10^6$, the ingested dose at 48 h is reduced to 0.7 log). This scenario underscores the importance not only of a particular competitive outcome (e.g., MPD_1 is limited to 5 log) but also the dependency of public health outcomes to the path taken in community-level dynamics.

The simulated dynamics become considerably more complex for Scenario 5, in which r_1 and r_2 vary stochastically between 1- and 3-h generation times (with 90% correlation between the growth rates). Even in this simple case where the remaining competition model parameters are fixed at their values under Scenario 1, the influence of stochastic growth rates on the community-level dynamics is reflected in the broad spread in the distributions for the risk assessment model outputs. Using Monte Carlo simulation methods to assess this scenario, the ingested dose of *E. coli* O157:H7 at 48 h has a median value of 4.2 log cfu (with a 95% credible interval (CI) of 2.1–5.8 log cfu), and the probability of illness has a median value of 0.20 (with a 95% CI of 0.003–0.62). (The 95% CI is the span between the 2.5th percentile and the 97.5th percentile of the Monte Carlo simulation output distribution.) Note that this two-order-of-magnitude span in the 95% CI for the probability of illness is obtained while varying only the microbial growth rates in the simulation. Consider, for example, that under Scenario 5, the duration of community growth is fixed at 48 h. In reality, however, time for growth is also variable and uncertain.

While the preceding scenario is hypothetical, it suggests that integrating complex microbial community dynamics into risk assessment can result in a large degree of variability and uncertainty. In the absence of ideal data from comprehensive, community-level predictive microbiological studies, food safety risk assessment can proceed nonetheless by characterizing the uncertainty about pathogen population growth dynamics under natural conditions based on a provisional understanding informed by available empirical data and ecological theory. Sensitivity analysis can then be performed to assess the importance of this uncertainty relative to other factors on the overall uncertainty in risk assessment endpoints.

Because the maximum growth of *E. coli* O157:H7 possible in ground beef depends on the variable and uncertain population of all microbes in the ground beef, it follows that the maximum population density is both variable and uncertain. One approach to modeling this variability and uncertainty in the absence of ideal data is presented by the draft risk assessment of *E. coli* O157:H7 in ground beef (Food Safety and Inspection Service, 2001), where a triangular distribution is used to model the variability in maximum pathogen density. The minimum is assumed to be 5 log, the maximum is assumed to be 10 log, and the most likely value is uncertain but can range uniformly from 5 to 10 log. In sensitivity analysis of the draft risk assessment model, the uncertainty related to the maximum potential population density was highly correlated with the uncertainty distribution for the density of *E. coli* O157:H7 in consumed servings.

4. Discussion

This paper presents a highly simplified model of complex microbial community dynamics. In order to gain additional insights into competitive interactions for experimental design or other purposes, the basic Lotka–Volterra competition model can be augmented in various ways for greater realism and generalizability. For example, one could also explore the influence of allowing the Lotka–Volterra model parameters and initial densities to vary jointly and stochastically as a function of environmental conditions and/or in density- or sequence-dependent fashion. Growth rates may be modeled as dependent on temperature, salinity, and pH. If control over the production and release of bacteriocins or other inhibitory substances by benign microorganisms that compete with pathogens is dependent on quorum sensing, then the competition model can be extended to describe such density-dependent dynamics. Similarly, Dens et al. (1999) discuss the potential to extend their model (Eq. (2)) to include effects such as the influence of competitors on lag phase duration and spatially heterogeneous food products. Circumstances also may arise in which

the contamination events for different organisms in a food product are likely to be sequential rather than concurrent due to different sources of contamination (e.g., enteric or environmental). Sequence dependency may confer what economists term ''first mover advantage'' in analyzing competition among business firms (Porter, 1990). Auger (1993) provides an example from the ecological literature of the influence of varying activity sequence on competitive outcomes (e.g., a shift from strong competition with one species in extinction towards weak competition with two coexisting species). Further, the basic two-species competition model can be generalized to account for any number of co-occurring populations and for a variety of interspecific interactions (e.g., parasitism and predation). Systems modeling approaches are generally used to account for complex ecological community dynamics. Bartell et al. (1992) provide some useful background on the integration of community-level interactions and effects into quantitative ecological risk assessment modeling.

Ecological theory suggests a wide ranging continuum of microbial community dynamics are possible. Specifically, it suggests that the path and outcome of competitive interactions may be highly sensitive to initial conditions and random variation in key factors such as growth rates and interspecific competition coefficients. Initially, experimental results of *E. coli* O157:H7 cultured in naturally contaminated ground beef appear incongruous with previous reports of pathogen inhibition by natural food microflora. These results are consistent with a simple competition model, however. Deviations from previous empirical food microbiology results do not necessarily imply that a study is defective but more likely that any particular study or group of studies provides an incomplete picture of the complex microbial ecology of foods. In general, the effects of competition observed in experimental and observational research may depend on the timing of the observations. For example, the effect of competition may be obscured if samples are drawn before the onset of inhibition, or peak levels of a microbial population may not be observed if samples are drawn after the onset of inhibition. Therefore, the full manifestation of competition is not obvious to the cross-sectional observer.

From a risk management perspective, it may or may not be possible or prudent to postpone food safety decisions until the results of comprehensive, community-level predictive microbiological studies are available. While this determination must be informed by science, it is ultimately a judgment call that depends on the context of the food safety decision and the distribution of risks, costs, and benefits associated with taking or delaying a decision. Contrary to conventional wisdom, we must keep in mind that new science may increase rather than reduce uncertainty about public health risks (Putnam and Graham, 1993). In particular, the sensitivity of the competition model to initial conditions is a distinguishing characteristic of chaotic systems and raises the possibility of irreducible uncertainty regarding the effect of microbial community dynamics in assessing the public health risks of foodborne pathogens. Findings such as the importance of the uncertainty about maximum population densities relative to the uncertainty regarding ingested doses of *E. coli* O157:H7 in ground beef suggest, however, a potential payoff to further research on microbial community-level dynamics in foods. The integration of well-designed experimental and surveillance studies, empirical data analysis, and modeling may fail to eliminate uncertainty but holds out the promise of providing food safety risk managers with effective strategies for coping with chaos.

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